20071025 APPLICATION#10/524815

Prepared by RICHARD A. HOUGHTLING, Ph.D.

Date of Search: 25 October 2007 at 13:50

Strategy:

(FILE 'HOME' ENTERED AT 13:51:10 ON 25 OCT 2007)

FILE 'CAPLUS' ENTERED AT 13:51:21 ON 25 OCT 2007

E TRICYCLIC ANTIDEPRESSANT

L1 18962 S E2

E ANTIDEPRESSANT

L2 30989 S E15 OR E19

L3 5108 S L1 (L) L2

E SPHINGOMYELINASE

L4 2706 S E27 OR E30

L5 957 S ACID (L) L4

L6 10 S L3 AND L4

E RINDERPEST

L7 327 S E39

E SWINE (L) FEVER

E FEVER

L8 1531 S E50 (L) E63

L9 0 S (L7 OR L8) AND L5

L10 0 S (L7 OR L8) AND L3

E CERAMIDE/CT

L11 9994 S CERAMIDE

L12 400 S L11 (L) ANTIBODY

E NEUTRALIZ????

E NEUTRALIZING/CT

L13 3632 S E100+ALL

L14 1 S (L13 AND L11) OR (L5 AND L13)

Update Info: CAPLUS
FILE COVERS 1907 - 25 Oct 2007 VOL 147 ISS 18
FILE LAST UPDATED: 24 Oct 2007 (20071024/ED)

Cost: 73.24

ANSWER SUMMARY

L6 ANSWER 1 OF 10 CAPLUS

Use of inhibitors of acid sphingomyelinase and of acid sphingomyelinase reaction products for the prophylaxis and treatment of infectious diseases; 2004:177956 CAPLUS

L6 ANSWER 2 OF 10 CAPLUS

Interactions of acid sphingomyelinase and lipid bilayers in the presence of the tricyclic antidepressant desipramine; 2004:114189 CAPLUS

L6 ANSWER 3 OF 10 CAPLUS

Antidepressant-induced lipidosis with special reference to tricyclic compounds; 2000:204886 CAPLUS

L6 ANSWER 4 OF 10 CAPLUS

Method and compositions for disrupting the epithelial barrier function; 1998:268334 CAPLUS

L6 ANSWER 5 OF 10 CAPLUS

The tricyclic antidepressant desipramine causes proteolytic degradation of lysosomal sphingomyelinase in human fibroblasts; 1994:548955 CAPLUS

L6 ANSWER 6 OF 10 CAPLUS

Effect of tricyclic antidepressants on lysosomal sphingomyelinase activity; 1989:185842 CAPLUS

L6 ANSWER 7 OF 10 CAPLUS

Acidic sphingomyelinase: relationship with antidepressant- induced desensitization of beta-adrenoceptors; 1988:400600 CAPLUS

L6 ANSWER 8 OF 10 CAPLUS

Modifications of sphingomyelin and phosphatidylcholine metabolism by tricyclic antidepressants and phenothiazines; 1986:102334 CAPLUS

L6 ANSWER 9 OF 10 CAPLUS

Effect of tricyclic antidepressants on sphingomyelinase and other sphingolipid hydrolases in C6 cultured glioma cells; 1983:447827 CAPLUS

L6 ANSWER 10 OF 10 CAPLUS

Tricyclic antidepressants induce sphingomyelinase deficiency in fibroblast and neuroblastoma cell cultures; 1982:449327 CAPLUS

L14 ANSWER 1 OF 1 CAPLUS

Use of inhibitors of acid sphingomyelinase and of acid sphingomyelinase reaction products for the prophylaxis and treatment of infectious diseases; 2004:177956 CAPLUS

COST IN U.S. DOLLARS	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	0.21	0.21

L6 ANSWER 1 OF 10 CAPLUS COPYRIGHT 2007 ACS on STN

Title

Use of inhibitors of acid sphingomyelinase and of acid sphingomyelinase reaction products for the prophylaxis and treatment of infectious diseases

Accession Number

2004:177956 CAPLUS Full-text

Document Number

140:193037

Author/Inventor

Gulbins, Erich

Patent Assignee/Corporate Source

Germany

Source

Ger. Offen., 10 pp. CODEN: GWXXBX

Document Type

Patent

Language

German

Family Accession Number Count

1

Patent Information

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
DE 10239531	A1	20040304	DE 2002-10239531	20020823
CA 2497582	A1	20040304	CA 2003-2497582	20030821
WO 2004017949	A2	20040304	WO 2003-EP9254	20030821
WO 2004017949	A3	20040429		
AU 2003255468	A1	20040311	AU 2003-255468	20030821
EP 1531826	A2	20050525	EP 2003-792402	20030821
CN 1688316	A	20051026	CN 2003-824405	20030821
JP 2006505527	T	20060216	JP 2004-530234	20030821
US 2005209219	A1	20050922	US 2005-524815	20050218

Abstract

The invention concerns the use of inhibitors of acid sphingomyelinase and/or of inhibitors of products (especially ceramide) catalyzed by reaction of this enzyme for prophylaxis and/or therapy of infectious diseases. Inhibitors include antibodies, especially neutralizing antibodies, and/or antidepressants, especially tricyclic and/or tetracyclic antidepressants.

L6 ANSWER 2 OF 10 CAPLUS COPYRIGHT 2007 ACS on STN

Title

Interactions of acid sphingomyelinase and lipid bilayers in the presence of the tricyclic antidepressant desipramine

Accession Number
2004:114189 CAPLUS Full-text
Document Number
140:314910
Author/Inventor
Koelzer, Melanie; Werth, Norbert; Sandhoff, Konrad
Patent Assignee/Corporate Source
Kekule-Institut fuer Organische Chemie und Biochemie, Universitaet Bonn, Bonn, D-53121, Germany
Cleaned CS
Kekule-Institut fuer Organische Chemie und Biochemie, Universitaet Bonn, Bonn, D-53121, Germany
Source
FEBS Letters (2004), 559(1-3), 96-98 CODEN: FEBLAL; ISSN: 0014-5793
Document Type
Journal
Language
English
Abstract
The tricyclic antidepressant desipramine causes a decrease in cellular acid sphingomyelinase (A-SMase, EC 3.1.4.12) activity when added to culture medium of human fibroblasts. This effect can be prevented by incubation of the cells with the protease inhibitor leupeptin, which suggests that desipramine induces proteolytic degradation of the lysosomal enzyme. By using surface plasmon resonance (SPR, Biacore) we were able to monitor the interactions of A-SMase and substrate-containing lipid bilayers immobilized on the surface of a Pioneer L1 sensor chip. SPR binding curves show that the enzyme hardly dissocs. from the lipid surface at acidic pH values. On the other hand, a drop in binding signals (resonance units, RU) of approx. 50% occurred after injection of 20 mM desipramine. Our findings indicate that desipramine interferes with the binding of A-SMase to the lipid bilayers and thereby displaces the enzyme from its membrane-bound substrate. The application of control substances suggests a key role for the cationic moiety of desipramine. We hypothesize that the displacement of the glycoprotein A-SMase from the inner membranes of late endosomes and lysosomes by desipramine renders it susceptible to proteolytic cleavage by lysosomal proteases.
Publisher
Elsevier Science B.V.
Reference Count
23 THERE ARE 23 CITED REFERENCES AVAILABLE FOR THIS RECORD ALL CITATIONS AVAILABLE IN THE RE FORMAT

L6 ANSWER 3 OF 10 CAPLUS COPYRIGHT 2007 ACS on STN
Title
Antidepressant-induced lipidosis with special reference to tricyclic compounds
Accession Number
2000:204886 CAPLUS Full-text
Document Number
133:83686
Author/Inventor
Xia, Zhenlei; Ying, Gu; Hansson, Ann Louise; Karlsson, Hakan; Xie, Yi; Bergstrand, Anders; DePierre, Joseph W.; Nassberger, Lennart
Patent Assignee/Corporate Source
Unit for Biochemical Toxicology, Department of Biochemistry, Wallenberg Laboratory, Stockholm University, Stockholm, S-106 91, Swed.
Cleaned CS
Stockholm University
Source
Progress in Neurobiology (Oxford) (2000), 60(6), 501-512 CODEN: PGNBA5; ISSN: 0301-0082
Document Type
Journal; General Review

Language
English
Abstract
A review with many refs. is given. Cationic amphiphilic drugs, in general, induce phospholipid disturbances. Tricyclic, as well as other antidepressants belong to this group. In exptl. animals, antidepressants induce lipid storage disorders in cells of most organs, a so-called generalized phospholipidosis. This disorder is conveniently detected by electron microscopic examination revealing myelin figures. Myelin figures or myeloid bodies are subcellular organelles containing unicentric lamellar layers. The lipidotic induction potency during in vivo is related to the apolarity of the compound Metabolism of phospholipids takes place within the cell continuously. Several underlying mechanisms may be responsible for the induction of the phospholipid disturbance. For instance, it was suggested that the compds. bind to phospholipids and such binding may alter the phospholipid's suitability as a substrate for phospholipases. Free TCA or metabolites thereof may also inhibit phospholipases directly, as was demonstrated for sphingomyelinase in glioma and neuroblastoma cells. Both these mechanisms might result in phospholipidosis. Interaction between drug and phospholipid bilayer was investigated by NMR technique. There seems to be large differences in the sensitivities amongst different organs. Steroid-producing cells of the adrenal cortex, testis, and ovaries are in particular susceptible to drug-induced lipidosis. The so-called foam cells are lung macrophages located in the interstitium which become densely packed with myelin figures during TCA exposure. It requires about 3-6 wk of treatment to develop this converted cell. In cell cultures however, phospholipidosis is demonstrated already after 24 h only. It appears that the cells that undergo TCA-induced lipidosis may recover after withdrawal of the drug. The time required to achieve complete recovery ranges from 3-4 wk to several months, depending on the organ affected. Little is known about the functional significance of lipidosis. Even if TCA and other antidepressants show other effects, it was not possible to exclusively relate it to phospholipidosis. However, few attempts were made to correlate the physiol. effects of TCAs in exptl. animals to the morphol. changes associated with phospholipidosis. There is an increasing evidence however, that cationic amphiphilic drugs may have effects on immune function, signal transduction, and receptor-mediated events, effects that to some extent might be related to disturbances in phospholipid metabolism
Publisher
Elsevier Science Ltd.
Reference Count
55 THERE ARE 55 CITED REFERENCES AVAILABLE FOR THIS RECORD ALL CITATIONS AVAILABLE IN THE RE FORMAT

L6 ANSWER 4 OF 10 CAPLUS COPYRIGHT 2007 ACS on STN
Title
Method and compositions for disrupting the epithelial barrier function
Accession Number
1998:268334 CAPLUS Full-text
Document Number
129:8587
Author/Inventor
Elias, Peter M.; Feingold, Kenneth R.; Holleran, Walter M.; Thornfeldt, Carl R.
Patent Assignee/Corporate Source
Regents of the University of California, USA; Cellegy Pharmaceuticals, Inc.
Source
PCT Int. Appl., 62 pp. CODEN: PIXXD2
Document Type
Patent
Language
English
Family Accession Number Count
2
Patent Information

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9817253	A1	19980430	WO 1997-US19343	19971022
AU 9749193	A	19980515	AU 1997-49193	19971022
US 6190894	B1	20010220	US 1998-58401	19980409
US 6562606	B1	20030513	US 2000-608568	20000630

Abstract

Epithelial barrier function is disrupted in a host in need of topical administration of a physiol. active substance by applying to the epithelium a barrier-disrupting amount of ≥1 agent selected from (1) inhibitors of synthesis of ceramides, acylceramides, glucosylceramides, sphingomyelins, fatty acids, or cholesterol; (2) degradation enzymes for ceramides, acylceramides, glucosylceramides, or sphingomyelins; (3) inhibitors of degradation of phospholipids, glycosphingolipids, glucosylceramides, acylceramides, or sphingomyelins; and (4) inhibitors and stimulators of metabolic enzymes of free fatty acids, ceramides, and cholesterol. Thus, a combination of 5-tetradecyloxy-2-furancarboxylic acid (an inhibitor of acetyl-CoA carboxylase which is the rate-limiting enzyme in free fatty acid synthesis) and β-chloroalanine (an inhibitor of serine palmitoyltransferase, the rate-limiting enzyme in ceramide synthesis) increased delivery of lidocaine through mouse stratum corneum by 8-fold in vivo and increased transepidermal water loss. Thus, a combination of 5-tetradecyloxy-2-furancarboxylic acid (an inhibitor of acetyl-CoA carboxylase which is the rate-limiting enzyme in free fatty acid synthesis) and β-chloroalanine (an inhibitor of serine palmitoyltransferase, the rate-limiting enzyme in ceramide synthesis) increased delivery of lidocaine through mouse stratum corneum by 8-fold in vivo and increased transepidermal water loss.

Reference Count

1 THERE ARE 1 CITED REFERENCES AVAILABLE FOR THIS RECORD ALL CITATIONS AVAILABLE IN THE RE FORMAT

L6 ANSWER 5 OF 10 CAPLUS COPYRIGHT 2007 ACS on STN

Title

The tricyclic antidepressant desipramine causes proteolytic degradation of lysosomal sphingomyelinase in human fibroblasts

Accession Number

1994:548955 CAPLUS Full-text

Document Number

121:148955

Author/Inventor

Hurwitz, Robert; Ferlinz, Klaus; Sandhoff, Konrad

Patent Assignee/Corporate Source

Inst. Organ. Chem. Biochem., Bonn, D-53121, Germany

Cleaned CS

Inst. Organ. Chem. Biochem.

Source

Biological Chemistry Hoppe-Seyler (1994), 375(7), 447-50 CODEN: BCHSEI; ISSN: 0177-3593

Document Type

Journal

Language

English

Abstract

The effect of the tricyclic antidepressant desipramine on the processing of lysosomal sphingomyelinase (EC 3.1.4.12) was investigated by pulse-chase studies on [35S]methionine labeled cultured human skin fibroblasts. Desipramine induced rapid intracellular degradation of mature acid sphingomyelinase when added to the cells in the micromolar range, concomitantly abolishing the enzyme activity. Pulse chase labeling revealed the disappearance of mature enzyme forms when fibroblasts were treated with 25 μM

desipramine. Incubation of cells with 25 μM leupeptin, an inhibitor of thiol proteases, 24h prior to desipramine intoxication prevented this drug-induced effect. From these results the authors conclude that desipramine and possibly also similarly acting tricyclic antidepressants induce proteolytic degradation of acid sphingomyelinase.

L6 ANSWER 6 OF 10 CAPLUS COPYRIGHT 2007 ACS on STN

Title

Effect of tricyclic antidepressants on lysosomal sphingomyelinase activity

Accession Number

1989:185842 CAPLUS Full-text

Document Number

110:185842

Author/Inventor

Baumann, Nicole; Carre, Jean Baptiste; Albouz, Samia; Hauw, Jean Jacques; Autran, Brigitte; Boutry, Jeanne-Marie; Masson, Martial; Maurin, Yves

Patent Assignee/Corporate Source

Lab. Neuropathol. Raymond Escourolle, Hop. Salpetriere, Paris, 75651, Fr.

Cleaned CS

Lab. Neuropathol. Raymond Escourolle, Hop. Salpetriere, Paris, 75651, Fr.

Source

NATO ASI Series, Series A: Life Sciences (1988), 150(Lipid Storage Disord.), 627-34 CODEN: NALSDJ; ISSN: 0258-1213

Document Type

Journal

Language

English

Abstract

The lysosomal acid sphingomyelinase of C6 glioma cells was inhibited by active tricyclic antidepressants at therapeutically relevant concns., whereas the hydroxylated derivs. were inactive. In contrast, no effect on neutral sphingomyelinase was observed In fibroblasts in the presence of [14C]sphingomyelin, desipramine increased the ratio of sphingomyelin/phosphatidylcholine. In C6 glioma cell cultures, the increase in the sphingomyelin/phosphatidylcho line ratio was correlated with a decrease in the stimulation of adenylate cyclase by isoproterenol and thus to the down-regulation of β-adrenergic receptors. However, this effect was not observed in rats treated chemical with desipramine. No modifications of lysosomal sphingomyelinase activity could be observed in the cerebral cortex of rats treated with desipramine under conditions to that modulated β-adrenergic receptors. The role of these results in the action of tricyclic antidepressants in inducing lipidosis is discussed.

L6 ANSWER 7 OF 10 CAPLUS COPYRIGHT 2007 ACS on STN

Title

Acidic sphingomyelinase: relationship with antidepressant- induced desensitization of beta-adrenoceptors

Accession Number

1988:400600 CAPLUS Full-text

Document Number

109:600

Author/Inventor

Carre, J. B.; Boutry, J. M.; Baumann, N.; Maurin, Y.

Patent Assignee/Corporate Source

Lab. Neurochim., Hop. Salpetriere, Paris, 75651, Fr.

Cleaned CS

Lab. Neurochim., Hop. Salpetriere, Paris, 75651, Fr.

Source

Life Sciences (1988), 42(7), 769-74 CODEN: LIFSAK; ISSN: 0024-3205

Document Type
Journal
Language
English
Abstract
Previous results indicate a dose-dependent decrease of lysosomal sphingomyelinase activity induced by tricyclic antidepressants in cell cultures. A possible association of this effect with the antidepressant-induced down-regulation of β-adrenoceptors was postulated. The authors report the determination of β-adrenoceptor binding sites and lysosomal sphingomyelinase activity in the cerebral cortex of rats treated chronically with desipramine (DMI) or with the potential antidepressant drug midalcipran (which is devoid of effects on β-adrenoceptors). The effect of midalcipran on lysosomal sphingomyelinase activity was also determined on C6 glioma cells. In C6 glioma cells, midalcipran did not decrease sphingomyelinase activity, at variance with the enzymic inhibition induced by DMT. In the rat cerebral cortex, neither DMI nor midalcipran modified sphingomyelinase activity. In agreement with previously reported effects, DMI induced β-adrenoceptor desensitization in the rat cerebral cortex, while midalcipran remained ineffective. In the rat cerebral cortex, the activity of lysosomal sphingomyelinase was not modulated by chronic treatment with antidepressant drugs, whatever their effect on β-adrenoceptor sites. Sphingomyelinase activity is not associated with the desensitization of β-adrenoceptors, taken as an index of the therapeutic action of antidepressants.

L6 ANSWER 8 OF 10 CAPLUS COPYRIGHT 2007 ACS on STN
Title
Modifications of sphingomyelin and phosphatidylcholine metabolism by tricyclic antidepressants and phenothiazines
Accession Number
1986:102334 CAPLUS Full-text
Document Number
104:102334
Author/Inventor
Albouz, S.; Le Saux, F.; Wenger, D.; Hauw, J. J.; Baumann, N.
Patent Assignee/Corporate Source
Fac. Pharm., Damas, Syria
Cleaned CS
Fac. Pharm., Damas, Syria
Source
Life Sciences (1986), 38(4), 357-63 CODEN: LIFSAK; ISSN: 0024-3205
Document Type
Journal
Language
English
Abstract
Phenothiazines and tricyclic antidepressants, when added to the culture medium, decreased lysosomal sphingomyelinase [9031-54-3] activity in C6 rat glioma cells and human fibroblasts. The effect of chlorpromazine [50-53-3] and desipramine [50-47-5] was concentration dependent, and was observed after 3 h of incubation with the drugs at 1-10 μM. In C6 glioma cell cultures, the decrease in sphingomyelinase activity was related to the clin. effectiveness of phenothiazines, tricyclic antidepressants and derivs. Incorporation of [choline-14C]sphingomyelin showed that the metabolic pathway implying the synthesis of phosphatidylcholine from the hydrolysis of sphingomyelin and/or transfer of phosphorylcholine to phosphatidylcholine was also partially reduced.

L6 ANSWER 9 OF 10 CAPLUS COPYRIGHT 2007 ACS on STN
Title
Effect of tricyclic antidepressants on sphingomyelinase and other sphingolipid hydrolases in C6 cultured

glioma cells

Accession Number

1983:447827 CAPLUS Full-text

Document Number

99:47827

Author/Inventor

Albouz, S.; Vanier, M. T.; Hauw, J. J.; Le Saux, F.; Boutry, J. M.; Baumann, N.

Patent Assignee/Corporate Source

Lab. Neurochim., INSERM, Paris, 75651, Fr.

Cleaned CS

Lab. Neurochim., INSERM, Paris, 75651, Fr.

Source

Neuroscience Letters (1983), 36(3), 311-15 CODEN: NELED5; ISSN: 0304-3940

Document Type

Journal

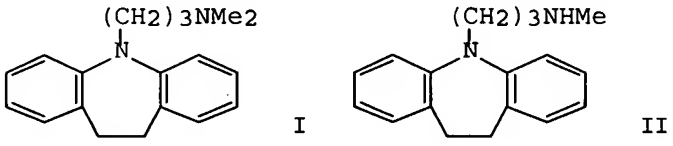
Language

English

Abstract

There was an early and important decrease in sphingomyelinase [9031-54-3] activity in C6 glioma cells cultured in the presence of the cationic amphiphilic drugs imipramine (I) [50-49-7] and desipramine (II) [50-47-5] at final concns. of 0.01 and 0.05 mM. The effect was dose-dependent and time-dependent and was observed before any lipid accumulation. Cerebroside β-glucosidase [37228-64-1] and cerebroside β-galactosidase [9027-89-8] had normal activities under the same exptl. conditions and thus there was no general effect on membrane-bound sphingolipid hydrolase. A decrease of sphingomyelinase activity has been previously reported for 2 amphiphilic compds., perhexiline maleate and AY 9944. These results suggest a potential function of sphingomyelinase in the mode of action of these drugs.

Graphics



I II

L6 ANSWER 10 OF 10 CAPLUS COPYRIGHT 2007 ACS on STN

Title

Tricyclic antidepressants induce sphingomyelinase deficiency in fibroblast and neuroblastoma cell cultures

Accession Number

1982:449327 CAPLUS Full-text

Document Number

97:49327

Author/Inventor

Albouz, S.; Hauw, J. J.; Berwald-Netter, Y.; Boutry, J. M.; Bourdon, R.; Baumann, N.

Patent Assignee/Corporate Source

Lab. Neurochim., Hop. Salpetriere, Paris, Fr.

Cleaned CS

Lab. Neurochim., Hop. Salpetriere, Paris, Fr.

Source

Biomedicine Express (1981), 35(7-8), 218-20 CODEN: BMEXBH; ISSN: 0300-0885

Document Type

Journal

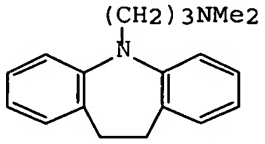
Language

English

Abstract

Tricyclic antidepressants (imipramine (I) [50-49-7] and desipramine [50-47-5]) gave rise to an important decrease of sphingomyelinase [9031-54-3] activity in murine neuroblastoma and human fibroblast cell cultures. It occurred within 1 to 2 h at a final concentration of 1 or 2 + 10-5 M in cell culture medium. Other lysosomal enzymes such as acid lipase, arylsulfatases A and B and hexosaminidases were not modified. Low level of sphingomyelinase activity may be related to the amphiphilic characteristics of the drugs; iminodibenzyl [494-19-9] which has the same tricyclic core but is devoid of the side chain necessary for amphiphilic properties had no effect. As iminodibenzyl has no therapeutic action, amphiphilicity may be requisite to antidepressant properties of tricyclic drugs.

Graphics



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L14 ANSWER 1 OF 1 CAPLUS COPYRIGHT 2007 ACS on STN

Title

Use of inhibitors of acid sphingomyelinase and of acid sphingomyelinase reaction products for the prophylaxis and treatment of infectious diseases

Accession Number

2004:177956 CAPLUS Full-text

Document Number

140:193037

Author/Inventor

Gulbins, Erich

Patent Assignee/Corporate Source

Germany

Source

Ger. Offen., 10 pp. CODEN: GWXXBX

Document Type

Patent

Language

German

Family Accession Number Count

1

Patent Information

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
DE 10239531	A1	20040304	DE 2002-10239531	20020823
CA 2497582	A1	20040304	CA 2003-2497582	20030821
WO 2004017949	A2	20040304	WO 2003-EP9254	20030821
WO 2004017949	A3	20040429		
AU 2003255468	A1	20040311	AU 2003-255468	20030821
EP 1531826	A2	20050525	EP 2003-792402	20030821
CN 1688316	A	20051026	CN 2003-824405	20030821

JP 2006505527	T	20060216	JP 2004-530234	20030821
US 2005209219	A1	20050922	US 2005-524815	20050218

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Special Queries
LinkOut
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NLM Mobile
NLM Catalog
NLM Gateway
TOXNET
Consumer Health
Clinical Alerts
ClinicalTrials.gov
PubMed Central

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- To save search indefinitely, click query # and select Save in My NCBI.
- To combine searches use #search, e.g., #2 AND #3 or click query # for more options.

Search	Most Recent Queries	Time	Result
#34	Search **neutralizing AND (#31) AND ceramide**	21:15:28	14
#33	Search **neutralizing AND (#31) AND (#2)**	21:13:28	1
#32	Search **neutralizing AND (#31)**	21:13:00	13326
#31	Search **antibody** Field: **MeSH Terms**	21:12:37	549124
#29	Search **(#10) AND (#11)**	19:59:56	7
#26	Search **(#4) AND (#11)**	19:54:50	14
#22	Search **(#6) AND (#19 OR #20 OR #21)**	19:51:52	4
#21	Search **(#18) AND (#16)** Field: **MeSH Terms**	19:49:53	101
#20	Search **(#11) AND (#16)** Field: **MeSH Terms**	19:49:47	5659
#19	Search **(#18) AND (#12)** Field: **MeSH Terms**	19:48:58	24816
#18	Search **opportunistic infection** Field: **MeSH Terms**	19:48:33	24816
#17	Search **(#12) AND (#16)** Field: **MeSH Terms**	19:48:04	4311
#16	Search **("associated conditions" OR "associated disease" OR "coexistent conditions" OR "concomittant disease" OR "concomittant conditions" OR "sequelae")** Field: **MeSH Terms**	19:47:34	95981
#14	Search **Infection control** Field: **MeSH Terms**	19:42:37	38256
#12	Search **Infection** Field: **MeSH Terms**	19:42:26	449736
#11	Search **Bacterial Infections and Mycoses** Field: **MeSH Terms**	19:40:49	881910
#10	Search **(#2) AND (#9)**	19:38:15	305
#9	Search **antagonists & inhibitors**	19:37:19	373294
#6	Search **Antidepressive Agents** Field: **MeSH Terms**	18:26:27	32973
#4	Search **Antidepressive Agents, Tricyclic** Field: **MeSH Terms**	18:24:00	7899
#3	Search **antidepressant** Field: **MeSH Terms**	18:21:34	32973
#2	Search **Sphingomyelin Phosphodiesterase** Field: **MeSH Terms** Sort by: **PublicationDate**	18:05:53	1767

Clear History